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شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

بالرسالة صفحات

لم ترد بالاصل

Ilizarov Technique in Treatment of Delayed and non Union of Tibial and Femoral Fractures

Thesis

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By

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INTRODUCTION

Bone repair

Bones heal in a unique and complex manner. In contrast to tissues that heal primarily with scar formation (skin, muscle, kidney, liver), and tissues that heal with a combination of more or less organized normal tissue as well as scar (nerves, and hollow organs), bones heal with new bone. However, bone healing is complex in that it usually requires the transformation of one tissue type into another (fibrous tissue to cartilage, and cartilage to bone. [1,2]

Fracture healing has been divided into primary fracture healing, and secondary fracture healing. Primary healing involves a direct attempt by the cortex to re-establish itself once it has become interrupted. Secondary healing involves responses in the periosteum, and external soft tissues with the subsequent formation of callus. [3]

Primary bone healing occurs only when there is anatomic restoration of the fracture fragments by rigid internal fixation, and when the stability of fracture reduction has been ensured by substantial decrease in the inter-fragmentary strain.[3]

The natural healing process includes the following stages:

1. The fracture

The fracture injures local marrow, periosteum, and adjacent soft tissues, as well as the living bone itself. As a result, some cells in these tissues die while others are sensitized so that they can respond better to special local and systemic messengers and stimuli. Also, the injury releases local biochemical and biophysical messengers that make the cells respond and help to determine how these cells may respond. These

messengers include mitogens, that make local precursor cells proliferate and messengers that guide the differentiation and organization of daughter cells. [4]

Essential parts of the first stage biologic responses finish within seven days after injury. Yet a clinician may need to wait for months for roentgenograms to show if inadequacies occurred during the original groundwork. [4]

2. The granulation tissue stage

Cells in the sensitized and stimulated local mediator mechanisms begin to produce new cells that differentiate and organize to produce new vessels, fibroblasts, intercellular materials, supporting cells and other cells in the space between fracture fragments. Macrophages, giant cells, and other wandering cells appear in the granulation tissue to invade and remove the dead tissues. Some osteoclasts usually appear at this time and erode some of the fracture surfaces. [4,5]

3. The callus

It is a unique tissue composed of fibrous tissue, cartilaginous, and bony elements. [1]

Further cell proliferation, differentiation, and organization begins to create new fibroblasts, chondroblasts, and osteoblasts in the granulation tissue. They synthesize the extracellular organic matrices of cartilage and woven bone. The cells come from two different origins. The first origin is the proliferation of determined osteoprogenitor cells in the periosteum and marrow. These cells directly produce membranous bone. The other origin is the inducible osteoprogenitor cells coming form pluripotent cells with chondrogenic potential that in due time are determined to undergo

subchondral bone formation. Mechanical, electrical and humoral factors may be able to induce the mesenchymal cells to differentiate to cartilage, and bone. These factors are known now as osteoinductors and the process is known as osteoinduction. Also, the cells need an appropriate environmental template, on which activated osteoprogenitor cells can produce bone, this is called osteoconduction. Osteoconduction also facilitates bone production and deposition in the appropriate three-dimensional array and enhances the ability of the regeneration process to bridge defects. Collagen and hydroxy apatite, are the proto type osteoconductive substances. [6]

A week or so later the newly synthesized matrices begin to mineralize. In humans, creation and mineralization of the callus after injury requires from four to more than 16 weeks. [4]

Callus formation occurs more slowly in adults and in compact bone than in children and in spongy bone. [4]

Mineralizaton is an extracellular process occurring near the osteoblasts, which exert a strong positive influence on the calcification process. Cells apparently control intracellular, as well as adjacent extracellular calcium and phosphate concentration. Calcium phosphate ions are taken up by the cells and then concentrated and stored in the mitochondria as amorphous non-crystalline calcium phosphate. These ions are then extruded from the cell into the matrix forming matrix vesicles; which are bounded by cell membrane. These may be the sites of initial calcification. Conversion of amorphous calcium into a collagen related crystalline form seems to occur extra-cellularly. [1]

The size of the soft callus formed and the relative portions of the fibrous, cartilagenous, and bony elements present at a given time are probably related to a variety of factors, of which only a few have been

identified. The first, is the amount of motion at the fracture site, more stable fixation generally results in smaller amounts of callus and less cartilage. The second is the degree of soft tissue and bony injury, including periosteal stripping from intact bone adjacent to the fracture site. The third is the availability of blood supply and capillary ingrowth (bringing nutrients and systemic hormones). [1]

Blood supply and rate of healing:

The long bones' vascular system is composed of three primary components, which are the principal nutrient artery, the metaphyseal arteries, and the periosteal arterioles. [7] Fig. 1

The principal nutrient artery traverses the diaphyseal cortex directly to enter the marrow cavity. There, it divides into arterioles, which are distributed to all areas of endosteal surface, to provide the major afferent supply of all diaphyseal cortex. [7,8]

The metaphyseal arteries are multiple, they supply the metaphysis at each end of the long bone. Terminal branches of the metaphyseal arteries enter the proximal and distal extremities of the medullary cavity to anastomose with terminal branches of the ascending and descending

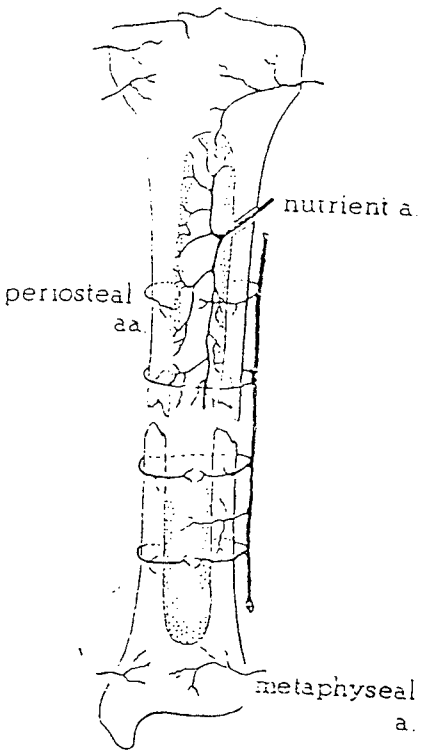

Fig.1 Bone circulation

medullary arteries. These anastomoses are very important because they permit metaphyseal arteries to maintain the medullary arterial supply when the principal nutrient artery has been ruptured by fracture or by surgery. [7,8]

The periosteal arterioles supply the outer third of the metaphyseal cortex. The periosteal vessels are derived from the main vessel of the limb and run transversely to the long axis of the bone; this is important because, when a bone is fractured, the nutrient vessels obviously are disrupted and, because they run longitudinally, the distal fragment is rendered avascular up to the point where the metaphyseal vessels enter the bone. However, because the periosteal vessels run transversely, the blood supply to the periosteum is maintained on both sides of the fracture site. The blood vessels in the viable periosteum hypertrophy and penetrate the cortex to re-establish the endosteal circulation. [9]

The periosteal arteries are richer in children than adults. They form together with the accompanying veins a continuos vascular layer around the bone. [9]

Relative participation of the three systems of vessels in callus formation:

It was found that when the periosteal vessels were the only source of nourishment to the callus, the periosteal circulation increased markedly leading to quicker formation of periosteal callus. When the periosteal blood flow is suppressed, if the bone marrow intact, an exuberant endosteal callus is formed but union is slightly delayed. When the metaphyseal-epiphyseal vessels were the only source of supply that remained the fracture united but in a slower rate and the union was only